AMENDMENTS TO THE SPECIFICATION:

Please add the following new headings before the paragraph beginning on line 4 of page 1:

--BACKGROUND OF THE INVENTION

Field of the Invention--

Please add the following new heading after the paragraph ending on line 10 of page 1:

--Description of related art--

Please add the following new heading after the paragraph ending on line 6 of page 3:

--BRIEF SUMMARY OF THE INVENTION--

Please add the following new headings and paragraphs after the paragraph ending on line 19 of page 3:

--BRIEF DESCRIPTION OF THE DRAWINGS

Figure 1 shows the major morphological changes on the mouse monocytes (control) obtained using compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9, the conversion resulting in dendritic cells (test compound).

Figure 2 shows PC12 cells (control) treated with the compound cholest-7-ene-3β,5α-diol-6β-N-[1-*N*1-(3-aminopropyl)butane-1,4-diamine] of Example 6 gradually change toward the formation of dendrites (test compound).

Figure 3 shows the induction of differentiation of the U-937 cells into dendritic cells by means of the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9 Photograph 1 shows the cells after treatment with 10 ng/ml of phorbol myristyl acetate (PMA) so as to make them adhere to the bottom of the culture dishes. Photograph 2 makes it possible, after treatment for 2 days with 1 nM of the compound of example 9, to observe the appearance of dendritic cells (CD). Photograph 3 shows, after treatment for 5 days, the increase in the size of the dendrites (up to 100 µm) and the formation of a large number of rosettes (R) consisting of an aggregate of entangled cells.

Figure 4 shows the production of cytokines(p70 subunit of IL-12, and IL-10) by the U-937 cells treated with the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of Example 9.

Figure 5 shows the activation of the T lymphocytes with the dendritic cells produced under the effect of the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9.

Figure 6 shows the cytotoxicity on TS/A in vitro: the tumoral cells were treated with variable doses of the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9.

Figure 7 shows the results of growth tests on tumors, where the test molecule is the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9, which is injected intradermally on the third day in the region of the tumor.

Figure 8 shows phase-contrast microscopy results of A59 cells treated with the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9. Photograph A shows the cells before treatment and photograph B after treatment; in photograph C, the cells were treated with the solvent vehicle; and in photograph D, it is observed that the treatment with the compound according to the invention leads to a massive appearance of vacuoles.

Figure 9 shows PC12 cells were treated with the compound cholest-7-ene-3β,5α-diol-6β-N-[1-*N*1-(3-aminopropyl)butane-1,4-diamine] of example 6 and the compound cholest-7-ene-3β,5α-diol-6β-N-[*N*,*N*'-bis(3-aminopropyl)butane-1,4-diamine] of example 7. The PC12 cells were incubated with each of these test compounds and with 10 ng/ml of NGF. The results are expressed as mean percentages of survival of the PC12 cells for three independent experiments.

Figure 10 shows purified motor neurones treated with the compound cholest-7-ene-3β,5α-diol-6β-N-[1-*N*1-(3-aminopropyl)butane-1,4-diamine] of example 6 and the compound cholest-7-ene-3β,5α-diol-6β-N-[*N*,*N*'-bis(3-aminopropyl)butane-1,4-diamine] of example 7. The purified motor neurones were incubated with 10 ng/ml of BDNF or with test compounds at a concentration of 100 nM.

Figure 11 shows A549 cells treated the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9 examined by electron microscopy. Figure 11.1 shows the "control" cells (magnification: 6000); figure 11.2 shows the cells treated with the test compound (magnification: 4000); figure 11.3 shows a magnification of the vesicles that can be seen in figure 11.2 (magnification: 25 000); figure 11.4 shows a detail of the content of an intracytoplasmic vacuole (magnification: 72 000).

The letters used in Figure 11 have the following meanings: N = nucleus; C = cytoplasm; M = mitochondria; Vi = intracytoplasmic vesicles; Ve = extracellular vesicles; MLB = multilamellar body.

Figure 12 shows A549 cells were treated with the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9. The actin skeleton of the treated cells was observed by labeling the cells with phalloidin-FITC. Photograph 1 of figure 12 shows the control A549 cells: the actin cytoskeleton holds the cells in a regular manner. Photograph 2 of figure 12 shows the appearance of refringent vesicles, which are surrounded by actin.

Figure 13 shows the observation of the tubulin skeleton by fluorescence microscopy of the A549 cells shown for Figure 12 . Photograph 1 shows an untreated control cell and photograph 2 shows a cell treated according to the process given in detail in example 21. In this case, the A549 cells are labeled with an anti-γ-tubulin antibody and developed with an anti-IgG-FITC antibody.

Figure 14 shows U937 cells treated with 10 nM of the compound the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9 for 24 hours. Photograph 1 corresponds to a "control" cell (magnification: 6000). Photograph 2 corresponds to a cell treated with the compound of example 9 (magnification: 4000): it is seen that the cell has changed shape and numerous intracytoplasmic vacuoles have appeared (referenced Vi in the photograph). The meanings of the reference letters in figure 14 are the same as those used for figure 11. In photograph 3 (magnification: 25 000), the presence of an extremely developed membrane network is observed; numerous cellular organites are

distinguished (numerous mitochondria, which are evidence of intense activity); multivesicular bodies (MVB) are also distinguished, the detail of which appears in photograph 4 of figure 14, this photograph showing an MVB in the course of secretion out of the cell (magnification: 72 000).

Figure 15 shows two immunofluorescence photographs of U937 cells expressing RCPG-HA and which were subjected to a treatment with the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9. These cells were fixed in order to be examined by fluorescence microscopy. The photographs of figure 15 show the expression of a membrane receptor of RCPG type in the cells that were treated with the compound according to the invention, whereas in the absence of treatment, the immunodetection is difficult to achieve.

Figure 16 shows The change in morphology of P19 cells treated with the compound cholest-7-ene-3β,5α-diol-6β-N-[1-*N*1-(3-aminopropyl)butane-1,4-diamine] of example 6 after 12 hours of treatment: the appearance of cellular extensions is observed, which change toward the formation of dendrites. Photograph 1 corresponds to a blank control and photograph 2 corresponds to the test compounds.

Figure 17 shows the efficacy of the compound cholestane-3β,5α-diol-6β-N-[2-ethylamino(1*H*-imidazol-4-yl)] of example 9 for an administration of this test compound remote from a tumor. Injection is performed on the third day after implantation of atumor, intradermally into the controlateral position relative to said implantation. In this experiment, a slowing-down of the

tumoral growth is observed relative to the control mice, for a dose of 1.7 μg/g of mouse.

DETAILED DESCRIPTION OF THE INVENTION--